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Camel milk attenuates the biochemical and morphological features of diabetic nephropathy: Inhibition of Smad1 and collagen Type IV synthesis

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Background: Fatty liver refers to a large spectrum of diseases characterized by excessive fat accumulation in the liver, which could be alcoholic or non-alcoholic in origin. Non-alcoholic fatty liver disease (NAFLD) is clinically important because it affects 25% of the population, with widespread pathological changes in the liver that range from simple non-progressive steatosis to non-alcoholic steatohepatitis (NASH). This can progress to cirrhosis, hepatocellular carcinoma, and liver failure with increased hepatic-related mortality.

Camel milk (CM) is gaining increasing recognition due to its beneficial effects in the control and prevention of multiple health probleMS. Recent studies have shown that CM has antihypertensive, anti-cancerous, hepatoprotective, and hypocholesterolemic effects. However, the effects of CM on high-fat, cholesterol-rich diet (HCD)-induced hepatic biochemical and structural changes, oxidative-antioxidative balance and glucose homeostasis have not been investigated.

Objectives: The current study aimed to investigate the effects of CM on the hepatic biochemical and cellular alterations induced by a high-fat, cholesterol-rich diet (HCD), specifically, non-alcoholic fatty liver disease (NAFLD).

Materials and Methods: Seventy male Wistar rats were divided into four groups: the Control (C) Group fed a standard diet; the Control + camel milk (CCM) Group fed a standard diet and CM, the Cholesterol (Ch) Group fed a HCD with no CM, and the Cholesterol + camel milk (ChM) Group fed a HCD and CM. The following parameters were investigated in the studied groups; basal, weekly random and final fasting blood glucose levels, intraperitoneal glucose tolerance test (GTT) and insulin tolerance test (ITT), serum insulin, serum lipids, liver functions, lipid peroxidation products, the antioxidant activity of catalase (CAT) and the levels of reduced glutathione (GSH). In addition, HOMA-IR as an index of insulin resistance (IR) and the histopathology of the hepatic tissue were assessed.

Results: The Ch Group developed features similar to those of non-alcoholic steatohepatitis (NASH), characterized by hepatic steatosis; inflammatory cellular infiltration in liver tissue; altered liver functions; and increased total cholesterol, triglycerides, low-density lipoprotein cholesterol, very-low-density lipoprotein cholesterol, AI, blood glucose, IR, and MDA levels. Additionally, feeding the HCD to animals in the Ch Group decreased CAT activity and the GSH and high-density lipoprotein (HDL) cholesterol levels. Camel milk intake for eight weeks decreased hepatic fat accumulation and inflammatory cellular infiltration, preserved liver function, increased the GSH levels and CAT activity, decreased the MDA levels, and ameliorated the changes in the lipid profile, AI, and IR in animals from the ChM Group.

Conclusions: The findings of the current study led us to conclude that camel milk markedly improved the biochemical and histopathological abnormalities induced by HCD, including hyperlipidemia, steatohepatitis, impaired liver function, and insulin resistance. The unique composition of CM that is rich in minerals, vitamins, insulin and insulin-like protein, and its antioxidant effects are likely responsible mechanism (s) for the altered metabolism and absorption of HCD. These findings support the reported health-promoting effects of CM and support its role in treating hyperlipidemia-associated chronic health probleMS resulting from unhealthy lifestyles and eating habits. Regular consumption of CM could provide a natural way to protect against NAFLD induced by a high-fat diet. However, large-scale clinical trials with large populations are still needed to confirm the results obtained from animal studies.

Key words: Camel milk; non-alcoholic fatty liver disease; steatohepatitis; high-fat diet; insulin resistance; hyperlipidemia; oxidative stress; rats.

Biography

Aida A Korish got her PhD degree from the College of Medicine- Alexandria University- Egypt at 2002. She is a Professor of physiology, College of medicine, Alexandria University. She is currently a visiting Associate Professor of Physiology, College of medicine, King Saud University. She has published more than 25 papers in reputed journals and has been serving as an global reviewer of may prestigious journals. She is interested in studying the blood biomarkers of renal diseases, the role of oxidant stress in renal diseases and the role of the alternative medicine in the treatment of chronic health probleMS.

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